Amendments to the Claims

Please amend the claims as follows:

Claims 1-11 (cancelled).

12. (Currently amended) An Anti-CEA/NCA antibody which specifically interacts with a subdomain of CEA/NCA, wherein said subdomain is selected from the group consisting of sequences G_{30}YSWYK (SEQ ID NO:1), N_{42}RQII (SEQ ID NO:2), and Q_{80}ND (SEQ ID NO:25).

13. (Previously presented) The antibody of claim 12, wherein said antibody releases a CEA/NCA-imposed inhibition of differentiation and/or apoptosis in CEA/NCA-producing primary and/or secondary tumor cells.

14. (Currently amended) A method for selecting a peptide or peptide-derived mimetics which can modulate a differentiation-blocking activity associated with a subdomain of CEA/NCA in a malignant tumor, wherein said subdomain is selected from the group consisting of sequences G_{30}YSWYK (SEQ ID NO:1); N_{42}RQII (SEQ ID NO:2; Q_{80}ND (SEQ ID NO:25); sequences including epitopes of 3 to 6 amino acids in the N-terminal 107 amino acid domain; and sequences including epitopes of 3 to 6 amino acids in the internal A3B3 178 amino acid domain of CEA, wherein said peptide or peptide-derived mimetics is selected as a modulator of said differentiation-blocking activity, when a tumor cell incubated with said peptide or peptide-derived mimetics, displays a significantly modified differentiation status as compared to a tumor cell incubated in the absence thereof.

15. (Currently amended) Peptides and/or peptide-derived mimetics obtained by the method of claim 14, wherein said peptide and peptide-derived mimetics interacting with subdomains of CEA/NCA involved in the differentiation-blocking activity associated with malignant tumors, wherein said subdomains are selected from the group consisting of sequences G_{30}YSWYK (SEQ ID NO:1), N_{42}RQII (SEQ ID NO:2), and Q_{80}ND (SEQ ID NO:25).

16. (Previously presented) A shankless anchor, which comprises a GPI anchor of CEA without the external domains thereof, wherein said GPI anchor interferes with downstream targets of endogenous CEA/NCA molecules to inhibit a differentiation-blocking activity thereof when administered to a primary or secondary tumor cell.

17. (Previously presented) A method to restore endogenous integrin function, which comprises:
an administration of a monoclonal antibody (MAB) that reverses a CEA/NCA-induced change in integrin function; or an
administration of a peptide or peptide-derived-mimetic that mimics an effect of said MAB;
thereby inhibiting a differentiation-blocking activity of endogenous CEA/NCA molecules.

18. (Previously presented) The method of claim 17, wherein said integrin function includes integrins $\alpha_5\beta_1$ and $\alpha_v\beta_3$.

19. (Previously presented) A drug screen assay to select a pharmaceutical agent which is capable of inhibiting a differentiation-blocking activity of endogenous CEA/NCA molecules in a cell, which comprises, an incubation of said cell with a candidate agent, wherein said pharmaceutical agent is selected when said differentiation-blocking activity is significantly inhibited in the presence of said candidate agent as compared to in the absence thereof.

20. (Currently amended) A method for enhancing efficacy of a cytoxic drug by increasing the differentiation status of tumor cells and/or by enhancing bystander effect, whereby more differentiated tumor cells cause adjacent autonomous tumor cells to behave more as non-malignant or normal cells, said method comprising an incubation of said tumor cells with an agent which interferes with one of a subdomain of CEA/NCA selected from the group consisting of sequences G_{30}YSWYK (SEQ ID NO:1), N_{42}RQII (SEQ ID NO:2), and Q_{80}ND (SEQ ID NO:25), and an integrin selected from the group consisting of $\alpha_5\beta_1$ and $\alpha_v\beta_3$, thereby increasing

said differentiation status and enhancing said efficacy of said drug.

21. (Currently amended) The method of claim 14, wherein said subdomain is selected from the group consisting of sequences G_{30}YSWYK (SEQ ID NO:1), N_{42}RQII (SEQ ID NO:2), and Q_{80}ND (SEQ ID NO:25).

22. (Previously presented) The method of claim 19, wherein said cell is a rat L6 myoblast expressing CEA/NCA.

23. (Previously presented) The method of claim 19, wherein said cell is a human Caco-2 colonocyte which aberrantly expresses a high level of CEA/NCA, and wherein said inhibition of differentiation-blocking activity can be positively correlated with a restoring of normal cellular and tissue architecture of said Caco-2 cells, upon incubation with said pharmaceutical agent.

24. (Currently amended) The method of claim 20, wherein said agent is selected from the group consisting of:

a) anti-CEA/NCA antibodies which specifically interact with a subdomain of CEA/NCA selected from sequences G_{30}YSWYK (SEQ ID NO:1), N_{42}RQII (SEQ ID NO:2), and Q_{80}ND (SEQ ID NO:25);

b) a peptide having a sequence selected from G_{30}YSWYK (SEQ ID NO:1), N_{42}RQII (SEQ ID NO:2), and Q_{80}ND (SEQ ID NO:25);

c) a peptide mimetic of b);

d) an antisense of CEA/NCA; and

e) a shankless anchor of CEA/NCA comprising a GPI anchor of CEA without the external domains thereof.

25. (Currently amended) A method of relieving a CEA/NCA-imposed inhibition of differentiation and/or apoptosis comprising an incubation of primary or secondary tumor cells with an agent which disrupts one of an interaction between CEA/NCA subdomains having sequences selected from G_{30}YSWYK (SEQ ID NO:1), N_{42}RQII (SEQ ID NO:2), and Q_{80}ND

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(SEQ ID NO:25), and a functional interaction between said subdomains and integrin $\alpha_5\beta_1$ and $\alpha_v\beta_3$.